Submasseteric Abscess Caused by *Mycoplasma salivarium* Infection[∇]

Andrea J. Grisold,¹* Martin Hoenigl,¹ Eva Leitner,¹ Klaus Jakse,² Gebhard Feierl,¹ Reinhard B. Raggam,¹ and Egon Marth¹

Institute of Hygiene, Microbiology and Environmental Medicine, Medical University of Graz, A-8010 Graz, Austria, and Elisabethinen Hospital, ENT-Department, A-8020 Graz, Austria²

Received 29 April 2008/Returned for modification 3 July 2008/Accepted 19 August 2008

Mycoplasma salivarium preferentially resides in the human oral cavity. Unlike other Mycoplasma species, M. salivarium has not been regarded as a pathogen, although one case of M. salivarium-caused arthritis in a patient with hypogammaglobulinemia has been reported. We describe the first case of submasseteric abscess caused by M. salivarium.

CASE REPORT

An 84-year-old woman presented with a several-week history of left buccal pain and swelling, trismus, and increasing difficulties in swallowing. She had a complicated past medical history of pulmonary embolism 2 months before admission, diabetes mellitus type 2, and arterial hypertension. Physical examination of the afebrile patient revealed nothing remarkable except for painful swelling of the left masseteric area with tenderness on palpation and trismus. Blood tests revealed an elevated white cell count of 29.3×10^9 /liter with 93.6% neutrophils, an elevated C-reactive protein level of 305 mg/liter, and a hemoglobin level of 9.6 g/dl. The patient was started on empirical antibiotic therapy of amoxicillin-clavulanic acid (2.2) g intravenously three times daily). After 4 days, she developed dysarthria. Magnetic resonance imaging of the brain was negative for an acute ischemic lesion. Therefore, the extensive left buccal swelling was considered causative.

A computed tomography (CT) scan showed an abscess formation over the left ramus mandibulae of 5 cm by 2.8 cm by 6.7 cm in size, which seemed to involve the mandibular joint, and a diffuse inflammatory thickening of the left masseter muscle, compatible with myositis (Fig. 1). The CT scan also showed periodontal osteolysis at the second premolar of the left mandible of 1.4 cm in diameter, which could have been the etiological cause for abscess formation.

Puncture fluid from the left submasseteric abscess was obtained for cytopathological and microbiological testing. Neutrophilic granulocytes and macrophages, but no malignant cells, were identified by cytology. After 4 days of incubation in the microbiology laboratory, very small (<1-mm), convex, limpid colonies were seen only on the Schaedler agar incubated under anaerobic conditions at 37°C, but the Gram stain gave a negative result. Therefore 16S rRNA gene analysis was carried out using universal eubacterial primers. Subsequently, a BLAST search of the obtained 16S rRNA gene sequence (665 bp) was performed using the taxonomy browser of the National

Center for Biotechnology Information (http://www.ncbi.nlm .nih.gov), and sequences were retrieved from GenBank. A homology of 100% was achieved for *Mycoplasma salivarium*, with the accession number AF125583.1. Subcultured onto A7 agar (bioMerieux, Marcy l'Etoile, France) under CO_2 -enriched atmosphere for 48 h, colonies showed a characteristic fried egg appearance under the light microscope at a magnification of $\times 10$. Resistance testing was performed with the Etest method (10). *M. salivarium* revealed susceptibility for ciprofloxacin, levofloxacin, moxifloxacin, and tetracycline; MICs obtained were $\leq 0.002~\mu g/ml$ (ciprofloxacin, levofloxacin, moxifloxacin) and $\leq 0.016~\mu g/ml$ (tetracycline). MICs for penicillin and amoxicillin-clavulanic acid were $\geq 32~\mu g/ml$ and $\geq 256~\mu g/ml$, respectively.

Left submasseteric abscess caused by *M. salivarium* was diagnosed. The antibiotic regimen was changed to moxifloxacin (400 mg orally daily) for a 3-week treatment course. After 1 week, a confined mass developed in the left oral vestibular region, which produced abundant pus after incision via an intraoral approach. Since *M. salivarium* was presumed to be the causative organism, no additional material was sent for microbiology testing. The patient's condition thereafter improved, the dysarthria resolved, and blood test results normalized, and the patient was discharged without further complaints.

Mycoplasma species are the smallest self-replicating bacteria and are generally commensal parasites in humans. Some species are real pathogens and are capable of causing a wide variety of diseases (2). The most common Mycoplasma spp. of the oral cavity are M. salivarium and M. orale (4). M. salivarium preferentially resides in dental plaques and gingival sulci (4). M. salivarium is usually found in 60 to 80% of throat specimens from adults and is also frequently found in inflamed tonsils (13). Normally, it is not regarded as a pathogen (12).

Reports of *M. salivarium* infections are rare, and only one case of a proven infection has been found in the literature. In 1983, *M. salivarium* caused arthritis in a patient suffering from hypogammaglobulinemia. Spurious contamination was not likely in this reported case, because isolation from synovial fluid of the patient's knee was made on three separate occa-

^{*} Corresponding author. Mailing address: Institute of Hygiene, Microbiology and Environmental Medicine, Medical University of Graz, Universitaetsplatz 4, A-8010 Graz, Austria. Phone: 43(316) 380-4383. Fax: 43(316) 380-9650. E-mail: andrea.grisold@meduni-graz.at.

[▽] Published ahead of print on 27 August 2008.

Vol. 46, 2008 CASE REPORTS 3861

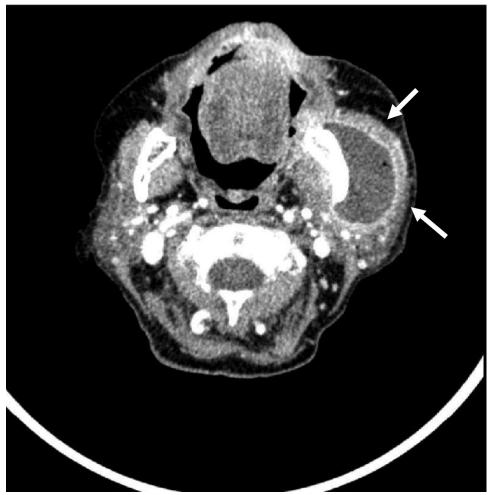


FIG. 1. CT scan image showing abscess formation over the left ramus mandibulae of 5 cm by 2.8 cm by 6.7 cm in size (upper arrow) and diffuse inflammatory thickening of the left masseter muscle (lower arrow).

sions months apart (12). Contamination of the bacterial culture in this described case of submasseteric abscess is extremely unlikely, because cultures of the submasseteric abscess material were incubated for a total of 14 days under aerobic and anaerobic conditions, but beside the extensive growth of *M. salivarium*, no additional growth of microorganisms could be observed.

M. salivarium is considered to participate etiologically in some cases of oral infections, including periodontal disease, based on the incidence and viable counts of the organism in the normal or pathological human oral cavity or gingival crevice, the antibody response to the organism, and the organism's biological and immunological activities (4, 11, 14). A variety of biochemical activities of M. salivarium are considered to facilitate both the attack of host tissues and escape from phagocytosis. M. salivarium stimulates human peripheral blood mononuclear cells and human gingival fibroblasts to induce interleukin-6 (IL-6), IL-8, IL-1β, and tumor necrosis factor alpha production (8, 11).

In a previous controlled study, it was shown that both the incidence of *M. salivarium* in the oral cavity and the metabolism-inhibitory (MI) antibody titers to the organism were

significantly higher in subjects with arthrosis temporomandibularis. Nine out of 14 subjects with arthrosis temporomandibularis were positive for metabolism-inhibitory antibodies to the organism (14). In another study, *M. salivarium* was detected in synovial fluids from 22 out of 33 patients with pain in or anterior disc replacement of the temporomandibular joint (15).

A recent study showed that synovial fluid samples from 2 out of 10 patients with osteoarthritis were positive for *M. salivarium*. Even higher detection rates were found for patients with traumatic osteoarthritis; five out of nine orthopedic patients with radiological changes consistent with at least mild osteoarthritis had infections caused by *M. salivarium* versus zero out of four patients in an orthopedic control group (6).

All these findings were quite surprising because, unlike other *Mycoplasma* spp., *M. salivarium* has not been regarded as a pathogen (12).

Infection by some *Mycoplasma* spp. has been suggested as a cofactor in the acceleration of immunodeficiency in human immunodeficiency virus (HIV)-infected patients (5). However, upon comparison, the detection rates of *M. salivarium*

3862 CASE REPORTS J. CLIN, MICROBIOL

and *M. orale* in HIV-positive and HIV-negative subjects were similar (3).

Submasseteric abscess is a rare infection between the masseter muscle and the mandible (7). Diagnosis of submasseteric abscess frequently eludes the clinician. The patient's history and clinical examination may be helpful, but as demonstrated in our case, CT scanning serves as an important additional diagnostic modality (7, 9). Infection usually arises from the posterior migration of organisms from an infected mandibular third molar (14).

Our CT scan findings suggest that osteolysis of a premolar was the most likely route of infection in the case described here. As in our case, the key to resolve the infection is surgical intervention to evacuate the pus, via either an intraoral approach or an extraoral incision for drainage (1, 9).

This is the first report of a submasseteric abscess caused by *M. salivarium* and one of the few reports of infection associated with this microorganism. We conclude that *M. salivarium* should be considered as a rare cause of oral and joint infections.

REFERENCES

- Barnard, N. A., and J. P. Magennis. 1992. Intra-masseteric actinomycosis: report of a case. Br. J. Oral Maxillofac. Surg. 30:190–191.
- Baseman, J. B., and J. G. Tully. 1997. Mycoplasmas: sophisticated, reemerging, and burdened by their notoriety. Emerg. Infect. Dis. 3:21–32.
- Chattin-Kacouris, B. R., K. Ishihara, T. Miura, K. Okuda, M. Ikeda, T. Ishikawa, and R. Rowland. 2002. Heat shock protein of Mycoplasma salivarium and Mycoplasma ovale strains isolated from HIV-seropositive patients. Bull. Tokyo Dent. Coll. 4:231–236.

- Engel, L. D., and G. E. Kenny. 1970. Mycoplasma salivarium in human gingival sulci. J. Periodontal Res. 5:163–171.
- Horowitz, S., J. Horowitz, L. Hou, E. Fuchs, B. Rager-Zisman, E. Jacobs, and M. Alkan. 1998. Antibodies to Mycoplasma fermentans in HIV-positive heterosexual patients: seroprevalence and association with AIDS. J. Infect. 36:79–84.
- Johnson, S. M., F. Bruckner, and D. Collins. 2007. Distribution of Mycoplasma pneumoniae and Mycoplasma salivarium in the synovial fluid of arthritis patients. J. Clin. Microbiol. 45:953–957.
- Jones, K. C., J. Silver, W. S. Millar, and L. Mandel. 2003. Chronic submasseteric abscess: anatomic, radiologic, and pathologic features. AJNR Am. J. Neuroradiol. 24:1159–1163.
- Kita, M., Y. Ohmoto, Y. Hirai, N. Yamaguchi, and J. Imanishi. 1992. Induction of cytokines in human peripheral blood mononuclear cells by mycoplasmas. Microbiol. Immunol. 36:507–516.
- Mandel, L. 1996. Diagnosing protracted submasseteric abscess: the role of computed tomography. J. Am. Dent. Assoc. 127:1646–1650.
- Ngan, C. C., T. Lim, C. M. Choo, G. L. Toh, and Y. S. Lim. 2004. Susceptibility testing of Singapore strains of Mycoplasma hominis to tetracycline, gatifloxacin, moxifloxacin, ciprofloxacin, clindamycin, and azithromycin by the Etest method. Diagn. Microbiol. Infect. Dis. 48:207–210.
- Shibata, K. I., A. Hasebe, T. Saski, and T. Watanabe. 1997. Mycoplasma salivarium induces interleukin-6 and interleukin-8 in human gingival fibroblasts. FEMS Immunol. Med. Microbiol. 19:275–283.
- So, A. K. L., P. M. Furr, D. Taylor-Robinson, and A. D. B. Webster. 1983. Arthritis caused by Mycoplasma salivarium in hypogammaglobulinaemia. Br. Med. J. 286:762–763.
- Tully, J. G. 1993. Current status of the mollicute flora of humans. Clin. Infect. Dis. 17:2–9.
- Watanabe, T., M. Matsuura, and K. Seto. 1986. Enumeration, isolation, and species identification of mycoplasmas in saliva sampled from the normal and pathological human oral cavity and antibody response to an oral mycoplasma (*Mycoplasma salivarium*). J. Clin. Microbiol. 23:1034–1038.
- Watanabe, T., K. I. Shibata, T. Yoshikawa, L. Dong, A. Hasebe, H. Domon, T. Kobayashi, and Y. Totsuka. 1998. Detection of Mycoplasma salivarium and Mycoplasma fermentans in synovial fluids of temporomandibular joints of patients with disorders of the joints. FEMS Immunol. Med. Microbiol. 22: 241–246